CASE REPORTS

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Pulmonary Aspiration of a Milk/Cream Mixture

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PULMONARY aspiration of milk in an adult has not been previously described. We recently treated a patient who aspirated a milk/cream mixture during induction of general anesthesia.

Case Report

A 69-yr-old, 70-kg man underwent transhiatal esophagectomy for Barrett's esophagitis. He returned to the hospital 9 days after discharge with nausea and vomiting associated with increasing shortness of breath and marked dyspnea. A computed tomography scan of his chest showed a large, left-sided pleural effusion. A chest tube was placed, and creamy fluid was withdrawn. Because the chest tube continued to drain chyle, the patient was scheduled for surgical repair of the thoracic duct.

The patient was ordered to fast overnight before surgery. A mixture of heavy cream (30 ml) and whole milk (30 ml) was to be taken orally 3 h before the scheduled start of surgery, but because of nursing delays, the mixture was actually ingested only 30 min before induction of anesthesia.

The patient was premedicated with 2 mg intravenous midazolam and 10 mg intravenous metoclopramide. In the operating room, after preoxygenation, cricoid pressure was applied, and a rapid-sequence anesthetic induction was commenced with 350 mg thiopental, 120 mg succinylcholine, and 100 μg fentanyl, all administered intravenously. Laryngoscopy showed a large amount of creamy material in the pharynx. The oropharynx was suctioned, and the patient's airway was intubated with a double-lumen tube. Both lungs were then immediately suctioned, and approximately 5-10 ml creamy material was removed from each one. Fiberoptic bronchoscopy was performed, and each lung was vigorously suctioned until clear.

The patient underwent video-assisted thoracoscopic repair of the thoracic duct with general anesthesia consisting of 1% isoflurane and 100% O_2. He received a total of 900 ml intravenous crystalloid solution during the procedure. For operative exposure, the left lung was selectively collapsed. He tolerated one-lung ventilation with an oxygen saturation as measured by pulse oximetry (Sp_{O_2}) of 98-100% throughout the 2-h procedure. After completion of surgery, a chest drainage tube was inserted, and the left lung was re-expanded. The anesthetic was then discontinued, and the patient's trachea was extubated. At that time, his Sp_{O_2} was 99%, his end-tidal carbon dioxide was 43 mmHg, and his spontaneous respiratory rate was 18 breaths/min.

In the postanesthesia care unit, the patient developed inspiratory and expiratory stridor. Auscultation of the lungs revealed diffuse bilateral crackles and wheezing. His respiratory rate increased to 30-44 breaths/min, and his Sp_{O_2} decreased to 88-94% while breathing oxygen by mask. An arterial blood gas test showed a *p*H of 7.17, oxygen partial pressure of 107 mmHg, and carbon dioxide partial pressure of 64 mmHg. A chest radiogram showed diffuse bilateral infiltrates. Despite treatments with nebulized albuterol, racemic epinephrine, and solumedrol, oxygenation did not improve. The patient's trachea was intubated with an 8.0-mm ID endotracheal tube, and he was transferred to the intensive care unit.

In the intensive care unit, the patient's lungs were mechanically ventilated with a tidal volume of 800 ml at a rate of 12 breaths/min with a 5-cm positive end-expiratory pressure. Sp_{O_2} ranged from 97-100% with an inspired oxygen fraction of 1.0. For controlled ventilation, he was sedated with continuous intravenous infusions of fentanyl and propofol.

A pulmonary artery catheter showed moderately elevated pulmonary artery pressures. He was begun on inhaled 20 ppm nitric oxide. His pulmonary artery pressure gradually decreased, and the nitric oxide was discontinued on the second day. At that time, he was weaned from mechanical ventilation, his trachea was extubated, and he was transferred to a surgical ward.

He was discharged from the hospital on the seventh postoperative day without any obvious permanent pulmonary sequelae.

Discussion

Chylothorax, lymphatic fluid in the pleural space from damage to the thoracic duct, is a recognized complication of transhiatal esophagectomy.[1,2] Surgical intervention is necessary to repair injuries refractory to intercostal tube drainage and supportive measures.[3] To identify

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the site of injury, a 250-ml milk and heavy cream (50:50) mixture can be given orally before surgery.[4] Milky fluid drips from the open thoracic duct, which is identified during surgery and oversewn.

Although the oral administration of this milk/cream mixture before surgery has been practiced since 1985, we could find no published report of pulmonary aspiration of this material. Our patient was probably at increased risk for this complication for several reasons.

After transhiatal esophagectomy, there is a loss of esophageal sphincter tone. The incidence of regurgitation is very high; with or without anesthesia it approaches 30%.[5] After esophagectomy, it would probably be safer to administer the milk/cream mixture through a jejunostomy tube.[6]

For our patient, the actual interval between ingestion of the mixture and induction of general anesthesia was very short. Had the anesthesia team been aware of this, we would have delayed the start of the anesthetic by several hours. The safe interval to fast after eating or drinking dairy products is unknown. For infants, it is recommended that at least 3 h elapse between breast feeding and surgery.[7]

The severity of pneumonitis increases as the *p*H of the aspirated fluid decreases.[8] The material our patient aspirated had a *p*H of 7.0. However, his clinical course was very similar to that reported after aspiration of a clear liquid with a very low *p*H. The *p*H may not be as important after aspiration of proteinaceous material such as milk and cream. In rabbits, the severity of injury was the same after installation of human breast milk (*p*H 7.0) and acidified breast milk (*p*H 1.8) into the lungs.[9] This study found that circulating neutrophils were increased after aspiration of both the normal and the acidified breast milk. This suggests that leukoactivation in response to lung injury was similar in both groups. Neutrophils have been implicated in the pathogenesis of acute lung injury after acid aspiration.[10]

Our patient's lung damage may have been worsened because he was exposed to 100% O_2 for 2 h immediately after the incident. Hyperoxia increases inflammatory lung injury after acid aspiration.[11]

We report a case of pulmonary aspiration of a milk/cream mixture in an adult. The usual recommended steps for reducing aspiration risk (Sellick's maneuver, metoclopramide, an H2-receptor antagonist, and an oral antacid) are probably ineffective after transhiatal esophagectomy. Although our patient's clinical course resembled that which follows pulmonary acid aspiration, it is possible that exposure to 100% O_2 immediately after the incident further exacerbated the lung injury.

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